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13. ABSTRACT <i>(Maximum 200 words)</i> The purpose of this study was to determine if operative oxygen desaturation occurs during transport from the operating room to the postanesthesia care unit in ASA 1 and 2 lower extremity orthopedic spinal anesthesia patients who received sedation with intravenous midazolam. Spinal anesthesia is considered a safe and effective alternative to general anesthesia. However, when spinal anesthesia is combined with intravenous sedation, patients may experience transitory respiration depression or even death. Spinal anesthesia patients are transported by anesthesia care providers from the operating room to the post anesthesia care unit without oxygen or oximetry monitors. This practice posed questions regarding patient safety during a critical postoperative period. While oxygen desaturation has been found to occur during transport in general anesthesia patients, it has not been studied in spinal anesthesia patients. Clear transport guidelines were not available from professional or regulatory sources. This study was conducted at a military community hospital in the central United States. A nonprobability, convenience sample was selected from patients presenting for lower extremity orthopedic surgical procedures in which subarachnoid blockade was appropriate. A data collection tool was used to collect the following information: demographics, preoperative oxygen saturation (SpO ₂) and baseline vital signs; subarachnoid block medications, dose, and patient position during insertion; dermatome level of analgesia prior to removal of supplemental oxygen; patient temperature prior to transport; SpO ₂ prior to removal of supplemental oxygen; total surgery time; total dose of midazolam administered; length of time from dose of midazolam; lowest SpO ₂ during transport from the operating room to the post anesthesia care unit; and total transport time. Our study found no incidence of hypoxemia (SpO ₂ < 90%) in this patient sample. There was a statistically significant decrease ($p < 0.001$) in the SpO ₂ from 98.9 (+ 1.54, range 96-100) to 98.0 (+ 1.20, range 95-100). However, this decrease was not found to be clinically significant as 95% SpO ₂ is a normal reading in adult humans breathing room air. Body mass index, smoking history, and dermatomal level of analgesia did not have...			
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POSTOPERATIVE OXYGEN DESATURATION FOLLOWING
SPINAL ANESTHESIA

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A Cluster Research Study

Submitted in partial fulfillment
of the requirements for the degree of

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The University of Texas Health Science Center at Houston

School of Nursing

October, 1997

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Abstract

The purpose of this descriptive, prospective study was to determine if postoperative oxygen desaturation occurs during transport from the operating room to the postanesthesia care unit in ASA 1 and 2 lower extremity orthopedic spinal anesthesia patients who received sedation with intravenous midazolam. Spinal anesthesia is generally considered a safe and effective alternative to general anesthesia. However, when spinal anesthesia is combined with intravenous sedation, patients may experience transitory respiratory depression or even death. In our institution, spinal anesthesia patients are transported by anesthesia care providers from the operating room to the post anesthesia care unit without oxygen or oximetry monitors. This practice posed questions regarding patient safety during a critical postoperative period. While oxygen desaturation has been found to occur during transport in general anesthesia patients, it has not been studied in spinal anesthesia patients. Additionally, clear transport guidelines were not available from professional or regulatory sources.

This study was conducted at a military community hospital in the central United States. A nonprobability, convenience sample was selected from patients presenting for lower extremity orthopedic surgical procedures in which subarachnoid blockade was appropriate. A data collection tool was used to collect the following information: demographics, preoperative oxygen saturation (SpO_2) and baseline vital signs; subarachnoid block medications, dose, and patient position during insertion; dermatome level of analgesia prior to removal of supplemental oxygen; patient temperature prior to transport; SpO_2 prior to removal of supplemental oxygen; total surgery time; total dose of

midazolam administered; length of time from last dose of midazolam; lowest SpO₂ during transport from the operating room to the post anesthesia care unit; and total transport time.

Our study found no incidence of hypoxemia (SpO₂ < 90%) in this patient sample. There was a statistically significant decrease ($p \leq 0.001$) in the SpO₂ from 98.9 (± 1.54 , range 96-100) to 98.0 (± 1.20 , range 95-100). However, this decrease was not found to be clinically significant as 95% SpO₂ is a normal reading in adult humans breathing room air. Body mass index, smoking history, and dermatomal level of analgesia did not have statistical significance related to oxygen saturation in this sample.

The results of this study support the safety of transporting ASA 1 and 2 lower extremity orthopedic patients without supplemental oxygen from the operating room to the post anesthesia care unit. This may result in decreased costs by reducing the use of supplemental oxygen, supplies, and manpower. Additionally, this study may lend itself as a source for determining supplemental oxygen and transport guidelines for similar patients in perioperative settings.

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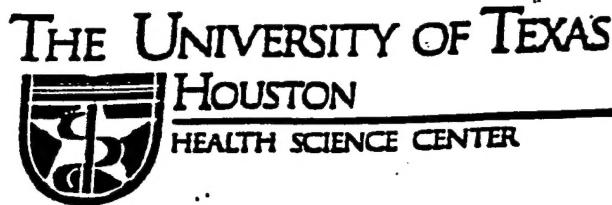
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The Committee for the
Protection of Human Subjects

NOTICE OF APPROVAL TO BEGIN RESEARCH

October 18, 1996

HSC-SN-96-075 - "Postoperative Oxygen Desaturation Following Spinal Anesthesia"
P.I.: Vicki J. Nichols, MAJ, AN, Student Nurse Anesthetist; et al

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APPROVAL DATE: October 18, 1996

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CHAIRPERSON: Anne Dougherty, MD

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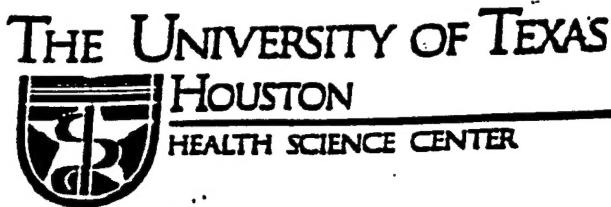
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The Committee for the Protection of Human Subjects

October 18, 1996

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CHAPTER I

Introduction

Spinal anesthesia is generally considered a safe and effective alternative to general anesthesia. It is not, however, without risk. When combined with intravenous sedation, patients may experience transitory respiratory depression and even death. Caplan, Ward, Posner, and Cheney (1988) reviewed approximately 900 closed claim cases for major anesthetic mishaps. They described 14 cases of healthy patients who experienced sudden cardiac collapse during spinal anesthesia with sedation for relatively minor surgical procedures. They hypothesized that depression of ventilation contributed to respiratory arrest which was followed by cardiopulmonary arrest.

Midazolam, which is often administered for sedation and amnesia during spinal anesthesia, has a high margin of safety and is not associated with respiratory or cardiovascular depression (Hobbs, Rall, & Verdoorn, 1996). However, when midazolam is combined with spinal anesthesia, ventilatory depression may result (Gauthier, Dyck, Chung, Romanelli & Chapman, 1992; Manara, Smith, & Nixon, 1989; Smith & Crul, 1989).

A possible explanation of the respiratory depression noted in sedated spinal anesthesia patients may be the interruption of normal feedback mechanisms which influence ventilation. These mechanisms include voluntary control, central chemoreceptors, peripheral chemoreceptors, and joint/chest wall proprioceptors. These mechanisms are further discussed in the upcoming conceptual framework section. In

general, if feedback from these mechanisms are blocked by the effects of midazolam or spinal anesthesia, ventilation may be altered. With the routine use of portable pulse oximeters in the late 1980s, investigators found that general anesthesia patients experience desaturation during transport from the operating room to the post anesthesia care unit (Smith & Crul, 1988; Tyler, Tantisira, Winter & Motoyama, 1985). These studies led to a change in practice, and most general anesthesia patients currently are transported with oxygen from the operating room to the post anesthesia care unit (J. Sherner, Anesthesia Nurse Consultant to the Army Surgeon General, personal communication, July 1996). Oxygen saturation of spinal anesthesia patients has been studied perioperatively but not during transport (Askrog, Smith, & Eckenhoff, 1964; Steinbrook, Concepcion, & Topulos, 1988; Yamakage, Namiki, Tsuchida, & Iwasaki, 1992). Our study filled a gap in the literature by investigating the oxygen saturation of sedated spinal anesthesia patients during transport from the operating room to the post anesthesia care unit.

Statement of the Problem

Our study examined the incidence of oxygen desaturation in adults during transport between the operating room and the post anesthesia care unit after spinal anesthesia with midazolam sedation.

Conceptual Framework

A physiological model of ventilation served as the conceptual framework for this study. Breathing is initiated in the central nervous system automatically; conscious thought is not required. The intrinsic rhythmicity of breathing is modified by neural and

chemical input to the respiratory control centers. These neural and chemical feedback systems will be explained further after a brief review of anatomy.

The respiratory centers, located in the brain stem, regulate the rate and depth of breathing (Levitsky, 1995). As shown in Figure 1, the respiratory centers consist of four groups of neurons in the medulla: the dorsal group, ventral group, apneustic center, and pneumotaxic center. While the exact mechanism of ventilation is not known, these areas act in concert to generate a rhythmic respiratory pattern.

Dorsal respiratory group. The dorsal respiratory group discharges mainly during inspiration. Its efferent axons stimulate the phrenic nerve which innervates the diaphragm.

Ventral respiratory group. The ventral respiratory group contains inspiratory and expiratory neurons which innervate laryngeal, pharyngeal, intercostal, and abdominal muscles. The diaphragm also is innervated by the ventral group via the phrenic nerve.

Apneustic center. The apneustic center in the caudal pons terminates inspiration and initiates expiration.

Pneumotaxic center. The pneumotaxic center in the superior pons “fine tunes” the ventilatory pattern.

The respiratory centers do not operate independently; several feedback mechanisms provide input to ensure the metabolic needs of the body are met. Feedback mechanisms included in our conceptual framework include voluntary control, central chemoreceptors, peripheral chemoreceptors, and limb/chest wall proprioceptors.

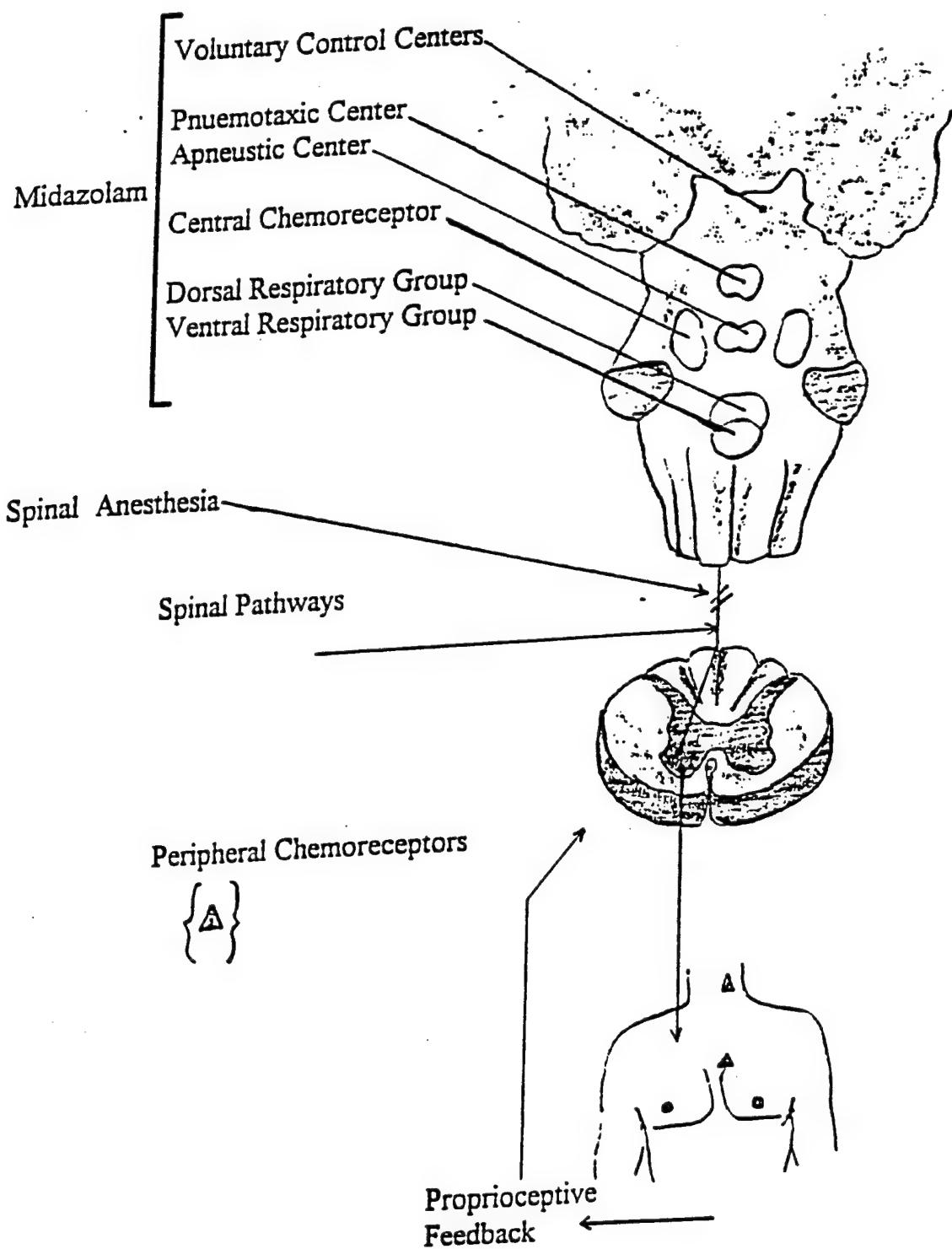


Figure 1. Anatomy of the respiratory control centers and feedback mechanisms.

Voluntary control. Although higher cortical centers in the central nervous system normally do not provide input into the normal pattern of respiration, voluntary control can override the medullary centers. Voluntary maximal inspiration produces the largest minute ventilation (V_E) attainable.

Central chemoreceptors. Central chemoreceptors, located in the ventrolateral surface of the medulla, monitor carbon dioxide (CO_2) and hydrogen ion concentration (pH). Stimulated mainly by an increase in CO_2 , these receptors exert their influence through afferent fibers to increase ventilation.

Peripheral chemoreceptors. Peripheral chemoreceptors, located in the carotid and aortic bodies, monitor arterial O_2 , CO_2 , and pH. Increases in ventilation are stimulated by hypoxemia and acidosis.

Afferent proprioceptors. Afferent proprioceptors, which are blocked during spinal anesthesia, stimulate the respiratory center to increase the rate and depth of inspiration in response to limb movements (Levitzky, 1995; Nadel, Waisbren, & Whiting, 1990). An increase in ventilation occurs prior to activation of the chemoreceptors by CO_2 and lactic acid. While the exact mechanism is not well understood, feedback from limb proprioceptors may explain the increase in ventilation which occurs at the onset of movement.

Sedative medications may compromise respiratory function by several mechanisms (Stevens & White, 1994). These mechanisms include direct depression of the central respiratory control centers (pneumotaxic center, apneustic center, dorsal respiratory group, and ventral respiratory group) and depression of ventilatory responses to hypoxia

and/or hypercarbia (central chemoreceptors). Additionally, decreases in oropharyngeal and head and neck musculature tone may result in upper airway obstruction, and reduction of respiratory muscle tone may result in decreased ventilatory effort (voluntary control) and increased ventilation-perfusion (V/Q) mismatch.

Spinal anesthesia interrupts afferent and efferent communication between the central nervous system and the periphery. Spinal anesthesia patients lose the contribution of joint/chest wall proprioceptive input into ventilation.

In summary, sedated spinal anesthesia patients may be at risk for oxygen desaturation due to interference with neural and chemical feedback into the respiratory control center. Spinal anesthesia blocks joint/chest wall proprioceptor input to the respiratory center. Midazolam sedation impedes the respiratory center's response to hypoxia and decreases oropharyngeal and head and neck musculature tone possibly resulting in upper airway obstruction, and reduction of respiratory muscle tone. A schematic diagram of the study variables is illustrated in Figure 2.

Purpose

The purpose of this study was to determine if, during transport from the operating room to the post anesthesia care unit, healthy adult patients with lower extremity orthopedic surgical sites experience oxygen desaturation following spinal anesthesia with midazolam sedation.

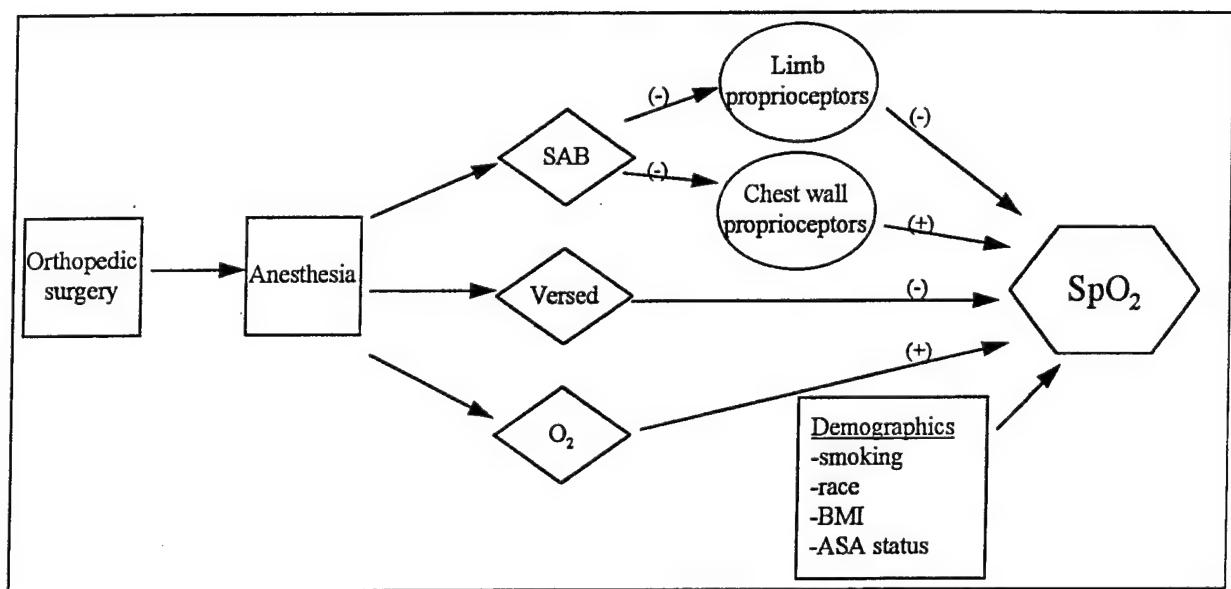


Figure 2. Conceptual framework. Schematic representation of study variables.

Definition of Terms

Oxygen saturation. Conceptual definition: "the statistical average of all oxygen bound to hemoglobin molecules relative to the total amount that can be bound" (Staub, 1993, p. 590). Operational definition: oxygen saturation as measured by pulse oximetry (SpO_2). Clinically significant desaturation is defined as $\text{SpO}_2 < 90\%$.

Sedation. Conceptual definition: depression of the central nervous system such that calming or drowsiness is produced (Hobbs, et al., 1996). Operational definition: intravenous injection of midazolam 50 - 100 $\mu\text{g}/\text{kg}$, administered in 10 $\mu\text{g}/\text{kg}$ increments until adequate sedation is achieved. To avoid oversedation, midazolam will not be administered if Verrill's sign (bisection of the pupil by the upper eyelid) or thickened speech is noted.

Spinal anesthesia. Conceptual definition: spinal anesthesia results in sympathetic block, sensory analgesia and motor block after injection of a local anesthetic solution into the subarachnoid space of the spinal column (Brown, 1994). Operational definition: injection of a hyperbaric local anesthetic, or a mixture of a hyperbaric local anesthetic and epinephrine, into the subarachnoid space of the spinal column to temporarily interrupt spinal cord nerve transmission.

Research Question

To what extent do adult postoperative spinal anesthesia patients experience a decrease in oxygen saturation during transport from the operating room to the post anesthesia care unit?

Significance of the Problem

Clear guidelines regarding the administration of oxygen and/or the use of a pulse oximeter to monitor SpO₂ during transport to the post anesthesia care unit after spinal anesthesia have not been elucidated in anesthesia literature. Review of the American Association of Nurse Anesthetists (AANA) monitoring standards, the Joint Commission on Accreditation of Healthcare Organizations standards, and anesthesia guidelines in the Army allow a great deal of latitude in assessing transport oxygen needs.

The AANA monitoring standards state that "adequacy of patient oxygenation shall be monitored continuously with pulse oximetry" (Aker & Rupp, 1994, p. 110). Specific oxygen monitoring guidelines for transport from the operating room are not written. Likewise, the Accreditation Manual for Hospitals (1996) provides a general guideline that anesthesia care providers determine oxygen requirements based upon individual patient assessment.

In Army hospitals, transportation policies vary from institution to institution. "The decision to administer oxygen or monitor oxygenation with a pulse oximeter is made by the individual anesthesia care provider. The Army does not have a policy governing transportation to the recovery room after spinal anesthesia" (J. Sherner, Anesthesia Nurse Consultant to the Army Surgeon General, personal communication, July 1996).

As stated earlier, oxygen saturation in spinal anesthesia patients has not been studied during transport from the operating room to the post anesthesia care unit. This study determined the incidence of oxygen desaturation in sedated spinal anesthesia

patients during transport. The results verify the safety of transporting sedated spinal anesthesia patients without oximetry monitors or oxygen (current practice).

Assumptions

1. Anesthesia care providers believe that healthy sedated spinal anesthesia patients do not desaturate during transport from the operating room to the post anesthesia care unit.
2. Patient desaturation during transport is undesirable.
3. Pulse oximetry is an accurate measurement of oxygen saturation.

Limitations

Pulse oximeters may give unreliable readings or cease functioning entirely when peripheral arterial pulsations are reduced or absent (Alexander, Gross, & Teller, 1989). Vasoconstriction and hypothermia can adversely affect pulse oximetry accuracy. Peripheral vasoconstriction may be due to coexisting peripheral vascular diseases, therefore, these patients were excluded from the study to avoid inaccurate pulse oximetry measurements. Because hypothermia can effect the accuracy of pulse oximetry readings, patients were not transported from the operating room with temperatures less than 34° C.

Another limitation is that patients received variable amounts of midazolam in order to achieve an adequate level of anxiolysis, amnesia, and sedation. To allow for patient differences in metabolism and liver enzyme function, setting a strict minimum or maximum dosage of midazolam was not possible. Additionally, patients were assured that their participation in the study would not effect the amount of medication they received. If a patient required a higher midazolam dose to achieve adequate sedation, amnesia and

anxiolysis, he/she received it. The total dose of midazolam administered and the time of the last dose prior to transport were documented.

A final limitation is the generalizability to other populations. This study was conducted in a U. S. Army medical facility. The convenience sample consisted mainly of healthy, young, ASA 1 and 2 males.

Spinal anesthesia combined with sedative medication did not result in oxygen desaturation during transport from the operating room to the post anesthesia care unit. This study supports the safety of transporting sedated ASA 1 and 2 spinal anesthesia patients without oximetry monitors or oxygen.

CHAPTER II

Review of Related Literature

To our knowledge, oxygen saturation during transfer from the operating room to the post anesthesia care unit has only been studied following general anesthesia. The review of literature was subdivided into the following categories: a) spinal anesthesia effects on ventilation with and without concomitant sedation, b) midazolam's effect on ventilation, and c) the incidence of desaturation during transport from the operating room to the post anesthesia care unit after general anesthesia.

Spinal Anesthesia Effects on Ventilation

Numerous studies have been done to evaluate the effects of spinal anesthesia on ventilation (Askrog et al., 1965; de Jong, 1965; Gauthier et al., 1992; McKenzie, Wishart, Dewar, Gray, & Smith, 1980; Steinbrook & Concepcion, 1991; Steinbrook et al., 1988; Yamakage et al., 1992). Table 1 outlines the multiple ventilatory parameters that have been used to describe the specific effects. Most of the studies used small sample sizes. Subjects received sedative premedication in three of the studies (Askrog et al., 1965; de Jong, 1965; McKenzie et al., 1980).

Askrog et al. (1965) demonstrated a 17 - 70% increase in respiratory dead space (V_D). Despite this finding, the PCO_2 did not change. The increased arterial - end-tidal carbon dioxide (a-ETCO₂) difference was evidence of a compensatory increase in minute ventilation (V_E). Other studies also reported an increase in V_E (Steinbrook & Concepcion, 1991; Steinbrook et al., 1988; Gauthier et al., 1992). Gauthier et al. reported an increase

Table 1

Effects of Spinal Anesthesia on Ventilatory Parameters

Researcher(s)	Block height	Sample size (n)	Results
Askrog et al. (1965)	T6 - T4	6	$\uparrow V_D$, $\uparrow a\text{-ETCO}_2$ difference, $\uparrow V_E^*$, V_T n.c.
de Jong (1965)	T10 - C5	32	PO_2 n.c., PCO_2 n.c.
McKenzie et al. (1980)	n.r.	49	PO_2 n.c., PCO_2 n.c.
Steinbrook et al. (1988)	T10 - T2	10	VC n.c., $\downarrow ETCO_2^*$
Steinbrook & Concepcion (1991)	T11 - T1	11	$\downarrow ETCO_2$, $\uparrow V_I^*$
Gauthier et al. (1992)	T8 - T3	19	$\uparrow V_T$, $\downarrow f$, $\uparrow V_E$, $\downarrow \%RC^*$
Yamakage et al. (1992)	T6 ± 1.4	20	$\uparrow \%RC$, $\uparrow SpO_2$, V_E n.c., SaO_2 n.c.

Note. * Parameter change not statistically significant; T = thoracic; C =

cervical; n.c. = no change; n.r. = not reported; V_I = minute inspiratory

ventilation; VC=vital capacity; f=frequency; SaO_2 =arterial oxygen saturation.

in V_E of 13% ($p < 0.01$). Each of these authors postulated that the increase in V_E was due to deafferentation of the chest wall. In contrast, Yamakage et al. (1992) found no change in V_E and suggested this was due to the lower level of spinal analgesia attained, as compared to other studies.

Spinal anesthesia does not change arterial oxygenation (de Jong, 1965; McKenzie et al., 1980; Yamakage et al., 1992). The single reported increase in SpO_2 was attributed to increased shunt to the cutaneous tissue via arteriovenous anastomoses (Yamakage et al., 1992) and was not further supported by published research.

The effects of spinal anesthesia on the percent of rib cage contribution to the tidal volume appears to be beneficial (Yamakage et al., 1992). However, Gauthier et al. (1992) reported a slight decrease in percent of rib cage contribution with spinal anesthesia. Both studies attribute any beneficial effects of increased percent rib cage involvement to deafferentation of the chest wall.

Spinal anesthesia in unpremedicated patients improves ventilation. However, spinal anesthesia in combination with midazolam has significant ventilatory depressant effects (Gauthier et al., 1992; Halim, Schneider, Claeys, & Camu, 1990; Manara et al., 1989; Smith & Crul, 1989).

Gauthier et al. (1992) reported moderate to profound sedation with midazolam combined with spinal anesthesia produced synergistic respiratory depression. In 19 healthy volunteers, V_T decreased 28% ($p < 0.01$) and frequency remained unchanged. Minute ventilation was decreased 19% ($p < 0.01$). The ventilatory depression was

attributed to altered mechanics due to spinal anesthesia and midazolam induced decrease respiratory drive.

Intraoperative administration of midazolam to patients with spinal anesthesia results in significant desaturation (Manara et al., 1989; Smith & Crul, 1989). Manara et al. studied 30 subjects undergoing transurethral resection of the prostate (TURP). Spinal analgesia levels of T11 - T6 were attained. Subjects were randomly assigned to two groups: Group A (n = 15) received supplemental oxygen by face mask and Group B (n = 15) breathed room air. Both groups received similar total dosages of midazolam. A significant decrease in oxygen saturation ($p < 0.001$) occurred in Group B. An elderly subject with ischemic heart disease developed electrocardiogram (ECG) changes highlighting the importance of this finding. The ECG changes were corrected with supplemental oxygen administration and the subject was withdrawn from the study. Smith and Crul planned to study 100 ASA 1 - 3 patients with spinal anesthesia and midazolam sedation. Midazolam was administered incrementally until subjects were "drowsy but arousable and able to communicate" (Smith & Crul, 1989, p. 206). Data collection was halted at 10 subjects due to an early trend demonstrating significant desaturation.

Spinal anesthesia has beneficial effects on ventilation. Intraoperative studies have demonstrated that synergistic ventilatory depression occurs when spinal anesthesia and midazolam sedation are combined. Due to prolonged effects of spinal anesthesia and midazolam, patients may be at increased risk for oxygen desaturation if transported without supplemental oxygen to the post anesthesia care unit.

Midazolam Effects on Ventilation

Pharmacodynamics. Midazolam is a short-acting water soluble benzodiazepine. The imadazole ring structure of midazolam is responsible for the drug's rapid metabolism and its stable state in aqueous solutions. Midazolam is a positive modulator of the gamma-aminobutyric acid (GABA) receptor in the central nervous system. The effects of midazolam on the central nervous system are dependent on the dose administered, the route of administration, and the presence or absence of other premedications (Hoffman La-Roche, 1994). Sedation after intravenous administration is achieved within 3 to 5 minutes. The time of onset is affected by total dose administered. Metabolism of midazolam is rapid with an elimination half time of 1 to 4 hours.

Sedative properties. Sedative doses of midazolam have little effect on minute ventilation and ventilatory response to CO₂ (Bailey et al., 1990; Curtis et al., 1989; Halim et al., 1990; Lindahl, 1990). However, at the larger dose required for hypnosis and unconsciousness, sensitivity to CO₂ is lost. As a result, the CO₂ response curve is shifted to the right and flattened (Figure 3). Sedative doses of midazolam slightly depress alveolar ventilation and cause respiratory acidosis as the result of a decrease in hypoxic rather than hypercapnic drive (Rall, 1990).

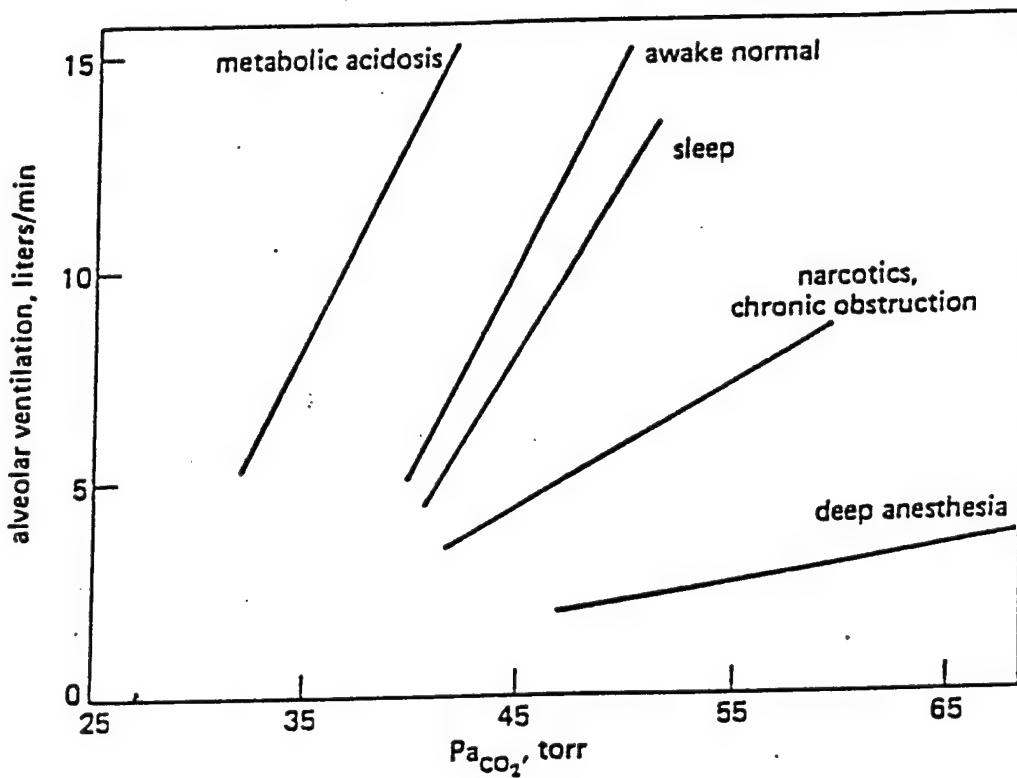


Figure 3. The effects of sleep, narcotics, chronic pulmonary obstructive disease, deep anesthesia, and metabolic acidosis on the ventilatory response to carbon dioxide.

From Pulmonary Physiology (4th ed.), by M. G. Levitsky, 1995, (p. 202). Copyright 1995 by McGraw-Hill, Inc. Reprinted with permission.

The ventilatory response to hypoxia is detected by the carotid bodies, located near the bifurcation of the carotid arteries. The arterial chemoreceptor system is composed of the carotid and aortic bodies. Interference of the arterial chemoreceptor system may lead to depression of the central respiratory center in response to hypoxia. The carotid bodies normally excite the respiratory control center through nervous stimulation. The hypoxic drive is the main ventilatory stimulant for elderly patients or patients with chronic obstructive pulmonary disease (Levitzky, 1995).

With sedative doses of midazolam, accessory muscles may improve ventilation by increasing the use of the intercostal muscles (Halim et al., 1990). However, during spinal or epidural anesthesia accessory muscles may be paralyzed, causing a decrease in chest wall compliance. As a result, the patient must work harder to breathe and normal ventilation may be compromised. The respiratory function of patients under centroneuraxis anesthesia combined with midazolam may be impaired.

Postoperative Transport

To our knowledge, oxygen saturation during transfer from the operating room to the post anesthesia care unit has only been studied following general anesthesia. Prior to the development of portable pulse oximeters, patients were routinely transported from the operating room to the post anesthesia care unit without oxygen after all types of anesthesia. The first study to use the new technology in assessing oxygenation after general anesthesia was completed by Tyler, et al. (1985). This descriptive study monitored 95 ASA class 1 or 2 patients during transport to the post anesthesia care unit

on room air. Thirty-five percent of their patients became hypoxic ($SpO_2 < 90\%$) and 12% became severely hypoxic ($SpO_2 < 85\%$).

A similar study compared the SpO_2 of 120 ASA 1 and 2 patients following elective surgery with general anesthesia (Smith & Crul, 1988). Sixty-four patients were transported to the post anesthesia care unit with two liters O_2 via nasal cannula, and 56 patients were transported on room air. Thirty-two percent of the room air group desaturated during transport ($SpO_2 < 90\%$) and 14.3% experienced severe hypoxia. None of the patients transported with oxygen desaturated.

The results of these and similar studies (Kataria, Harnik, Mitchard, Key, & Admed, 1988; Tait et al., 1990) led to a change of practice. Most anesthesia care providers currently transport general anesthesia patients to the post anesthesia care unit with oxygen.

Summary

In general, researchers have shown that oxygen desaturation is a potential complication in sedated spinal anesthesia patients. Numerous studies have demonstrated depressed ventilation parameters during spinal anesthesia with midazolam sedation. As Caplan et al. (1988) discussed, oxygen desaturation in spinal anesthesia patients may be the first sign of impending hemodynamic instability or cardiovascular collapse. Its prompt identification and treatment may prevent unexpected mortality and morbidity. While patients are closely monitored in the operating room and in the post anesthesia care unit, they are not routinely monitored or administered oxygen during the relatively short transport time between the two areas. Our study investigated the incidence of oxygen

desaturation during this traditionally unmonitored time period to determine whether supplemental oxygen or pulse oximetry monitoring is necessary for sedated ASA 1 and 2 spinal anesthesia patients.

CHAPTER III

Methodology

To determine the incidence of desaturation during transport from the operating room to the post anesthesia care unit after spinal anesthesia with sedation, a nonexperimental descriptive study was conducted. This chapter describes the population, sample, setting, instrumentation, procedure for data collection, protection of human subjects and the research design. Plans for data analysis, timeline, and budget complete the chapter.

Population, Sample, and Setting

Population and setting. The study was conducted at a 264-bed military health care facility, located in central Texas. Approximately 145,000 people receive healthcare from this community hospital (personal communication, J. W. Kirkpatrick, Hospital Commander, March 21, 1996). Potential study participants were selected from individuals receiving care at the facility and included active duty personnel, family members of active duty personnel, reservists on active duty, and retired military beneficiaries and their eligible family members.

Sample: Subjects for this descriptive study were selected from the population of patients scheduled for elective lower extremity orthopedic surgery who consented to spinal anesthesia. The method was a nonrandom, convenience sample.

Inclusion Criteria:

1. Male or female

2. Elective lower extremity orthopedic surgery patients
3. ASA 1 or 2 category
4. 18 to 60 years old
5. Understand the English language

Subjects were selected from a population of male and female lower extremity orthopedic surgery patients who consented to spinal anesthesia (inpatient or outpatient). Orthopedic surgical patients with surgical sites below the 10th thoracic dermatome were selected to control for the decreased diaphragmatic excursion, decreased chest wall compliance and splinting associated with thoracic and abdominal surgical sites (Stoelting & Dierdorf, 1993). All subjects included in the study were categorized as ASA 1 or 2 and between 18 and 60 years of age. The rationale for selecting ASA 1 and 2 elective surgery patients was to minimize the need for intraoperative medical interventions and to control for confounding variables which may alter the results of the investigation. The age parameters were selected based upon the assumption that groups outside of this range possess unique anatomy such as immature respiratory systems in the young or decreased lung capacities associated with aging. Additionally, subjects older than 60 years of age may have undiagnosed respiratory pathologies. Only English speaking patients were included in the study. This facilitated informed consent and patient understanding of the study.

Exclusion Criteria:

1. Pregnant females, up to six weeks postpartum
2. Body mass index greater than 35

3. Diagnosed pulmonary pathology
4. Patients with peripheral vascular disease
5. Incompetent patients

Exclusion criteria were selected to exclude patients whose physical status or pathology may affect their ventilatory status. Pregnant females were excluded due to their risk of rapid desaturation related to the combination of increased minute ventilation and decreased functional residual capacity (FRC) (Stoelting & Dierdorf, 1993). Obese patients with a BMI greater than 35 were excluded due to decreased FRC, increased risk of aspiration, and the restrictive effects of abdominal contents impeding diaphragmatic excursion. Diagnosed respiratory pathologies included asthmatics who reported an attack within the last 30 days, patients diagnosed with chronic obstructive lung disease, bronchitis, pulmonary embolism, or patients with an active upper respiratory tract infection. These patients were excluded to ensure accurate measurement of SpO₂ and to control confounding variables. Patients with peripheral vascular disease were excluded due to the inability of pulse oximetry to accurately measure oxygen saturation in this patient population. Finally, "if the patient's capacity to comprehend is impaired by language barrier, physical status, medications, unauthorized drugs or alcohol" (Mastropietro & Bruton-Maree, 1994, p. 257), they are considered incompetent. Incompetent patients were excluded due to their lack of understanding of the study and inability to follow directions during spinal anesthesia placement.

Sample Size: A convenience sample was used. All consenting patients that met the inclusion criteria during the data collection period were included in the sample. Power

analysis demonstrated that a sample of 50 patients was necessary for this study with a power of 0.80, a medium effect size (0.5) and a level of significance set at $p \leq 0.05$ (Cohen, 1988). A medium effect size was chosen based on recommendation of Cohen (1992) since this population has not been studied in the past. To ensure an adequate sample size after attrition, our goal was to oversample by 25 for a total of 75 patients.

Instrumentation

Pulse oximetry (SpO₂) provides a non-invasive means to measure oxygen saturation of arteriolar hemoglobin. This study used the Propaq pulse oximeter by Protocol Systems Inc. Propaq pulse oximeters are widely used and designed for post anesthesia monitoring and intra-hospital patient transport.

The Propaq pulse oximeter specifies a range of saturation from 0% to 100% (Protocol Systems Inc, 1989 - 1993). Probe accuracy is reported at $\pm 2\%$ (one standard deviation) at saturation ranges of 70% to 100% (28° to 42° C). Probe accuracy is unspecified from 0% to 70% oxygen saturation. To avoid threat of instrumentation error due to variation in equipment, two Propaq pulse oximeters were used for data collection. Consistency of data collection was maintained by using the same technique for acquiring the SpO₂ measurement, by placing the probe on a finger.

The verification and safety check was performed by a qualified biomedical technician to verify the accuracy of the instrument prior to the data collection process. Additionally, the Propaq 106 pulse oximeter is self calibrating.

Procedure for Data Collection

To ensure interrater reliability in assessing dermatomal level of spinal blockade, a standardized method of assessment was developed and the researchers were instructed in its use. Each researcher independently identified dermatomal levels on an anatomical model. Interrater reliability testing was obtained and differed by no more than one dermatome between researchers.

The researcher invited a potential research subject who met the inclusion/exclusion criteria to participate in the study. Written informed consent was obtained from those who agreed to participate (Appendix A).

1. An investigator obtained a baseline SpO₂ reading via the Propaq pulse oximeter prior to premedication, supplemental oxygen, or initiation of the subarachnoid block. This measurement was taken in the operating room or the holding area. The measurement was taken via a finger probe placed on a finger without nail polish. The measurement was recorded on the Data Collection Form (Appendix B).

2. In the operating room, after the initial SpO₂ reading was obtained, the investigator connected the subject to routine monitors. These monitors included SpaceLabs pulse oximeter, EKG monitor, temperature probe, precordial stethoscope, noninvasive blood pressure cuff, and other monitors deemed appropriate for the patient. Baseline vital signs were obtained and recorded on the Data Collection Form.

3. A peripheral intravenous line was inserted in the preoperative holding area and a fluid bolus of Lactated Ringers solution was administered. The patient was

premedicated with intravenous midazolam (up to 0.1 mg/kg) for anxiolysis.

4. Oxygen was administered via nasal cannula at a flow rate of 2 liters per minute during the surgical procedure.

5. Spinal anesthesia was induced at the lumbar level using a hyperbaric local anesthetic solution with or without epinephrine. The investigator noted medication(s), dose, patient position during insertion, and lumbar level of insertion on the Data Collection Form.

6. Intravenous midazolam for anxiolysis was provided as needed during the surgery. The dosage was individualized by the researcher for each patient. Vernil's sign (eyelids bisecting pupil) and/or slurred speech were indicators of the upper limit of sedation. The total dose of midazolam and time of last dose were recorded on the Data Collection Form.

7. Prior to transport to the post anesthesia care unit, the researcher ensured that the Propaq pulse oximeter's "low SpO₂" alarm was set at 90%. Additionally, the researcher verified that the gurney had a cylinder of oxygen containing more than 500 psi of oxygen with a nasal cannula and oxygen tubing attached.

8. At the completion of the surgery, the dermatomal level of analgesia was determined prior to transport to the post anesthesia care unit. The dermatomal level of analgesia was obtained via sharp/dull discrimination with a sterile 22 gauge needle along the midaxillary line bilaterally. The dermatome level was recorded on the Data Collection Form.

9. Prior to the removal of the patient's nasal cannula, the patient's SpO₂ was

measured using the Propaq pulse oximeter. From this point on, the oximeter probe remained on the patient's finger until the patient was received in the post anesthesia care unit. The initial Propaq pulse oximeter value was recorded as SpO₂I on the Data Collection Form. Additionally, the total surgery time (from initiation of the subarachnoid spinal anesthetic to patient placement on the gurney) was recorded. After recording the data, the nasal cannula was removed and the stopwatch was started to record the transport time between the operating room and the post anesthesia care unit.

10. With assistance, the patient was transferred to a gurney for transport to the post anesthesia care unit.

11. The researcher observed the Propaq portable pulse oximeter during transport. The lowest SpO₂ value noted during transport was recorded on the Data Collection Form as Low SpO₂.

12. If the patient's SpO₂ decreased to 90% during transport from the operating room to the post anesthesia care unit, supplemental oxygen was provided at a rate of 2 liters/minute via nasal cannula. The researcher recorded the 90% SpO₂ as a desaturation on the Data Collection Form, and recorded the time the desaturation occurred.

13. Upon final placement of the gurney in the post anesthesia care unit, the patient's SpO₂ was recorded and the stopwatch was stopped. Low SpO₂ during transport, final SpO₂, and total transport time were documented on the Data Collection Form.

Protection of Human Subjects

Permission to conduct the study was obtained from the Clinical Investigations Committee, Brooke Army Medical Center. Additionally, permission was obtained from

the Committee for Protection of Human Subjects of the University of Texas Health Science Center at Houston and the Institutional Review Board at Brooke Army Medical Center. Informed written consent was obtained from each participant. Subjects were assigned numbers for the purpose of maintaining anonymity and assisting with data analysis.

The procedure, potential complications and maintenance of confidentiality were discussed with the patients and concerned significant others during the preanesthetic interview. Subjects were informed that their refusal to participate would not alter or jeopardize their care. Confidentiality was maintained by not including subject names, hospital identification numbers, or social security numbers on data collection forms. A single document contained the subjects' names with their code numbers, so researchers could review records after data collection if needed. The code sheet was kept in a locked file drawer and destroyed upon completion of the study. The findings of the study were reported as summary data.

Study Design

This study used a non-experimental descriptive design. A threat to reliability was the use of a pulse oximeter to collect data on SaO_2 . Pulse oximeters may generate unreliable readings or cease functioning entirely when peripheral arterial pulsations are reduced or absent (Alexander et al., 1989). Vasoconstriction and hypothermia can adversely affect pulse oximetry accuracy. Peripheral vasoconstriction may be due to coexisting peripheral vascular diseases, patients with peripheral vascular diseases were excluded from the study to avoid inaccurate pulse oximetry measurements. Vital signs

were monitored throughout the surgery. Patients were not transported from the operating room with temperatures less than 34° C. The investigator prevented hypothermia by covering the patient with a blanket or warming device.

Inter-rater reliability may pose a threat to internal validity. Inter-rater reliability was controlled by each researcher demonstrating proper probe placement on the index finger and the ability to set the low-limit alarm for 90% SaO_2 prior to commencement of data collection. To minimize extraneous factors which may affect patient oxygenation, patients were screened to prevent inclusion of patients with pre-existing respiratory diseases or conditions which affect ventilation.

The nonrandom, convenience sample selection process poses threats to external validity and limits generalizability. Demographic data was analyzed to provide a data base for future research and to describe the sample as fully as possible.

Data Analysis

Descriptive information was reported using frequencies and measures of central tendency. Means and standard deviations were calculated for the following: age, weight, baseline SpO_2 , dermatomal level of analgesia upon leaving the operating room, total dose of midazolam, time from last dose of midazolam, SpO_2 prior to leaving the operating room, SpO_2 prior to transport, SpO_2 in the post anesthesia care unit, and transport time.

The mean and standard deviation were calculated for the SpO_2 measurement in the operating room, the lowest SpO_2 during transport, and the final SpO_2 reading in the post anesthesia care unit. Student's paired t-test was used to compare the SpO_2 mean scores. There were no incidences of desaturation to 90%. Statistical significance was set at $p <$

0.05. Linear regression analysis was used to evaluate the effects of multiple sample characteristics on the percent change in SpO₂.

Time Line

July 1, 1996	Clinical Investigations/Human Use Committee Meeting
July 15, 1996	Investigational Review Board Meeting
August 5, 1996	Executive Committee Review/Commander Approval
September 22, 1996	Establish inter-rater reliability
October 1996	University of Texas Health Science Center at Houston's Clinical Investigations/Human Use Committee
November 25, 1996	Data collection began
April 27, 1997	Data collection ended. Data analysis began
July 31, 1997	Completion of final draft
October 3, 1997	Thesis defense
October 31, 1997	UTHSSC submission

Budget

The equipment and supplies needed for this study were routinely used for patient care. No additional supplies were required for the study.

a. Travel for presentation(s).....	\$5,000
b. Poster for presentation(s).....	1,000
c. Manuscript binding.....	200
d. Stopwatches (3 @ \$15.00 each, tax).....	50

e. Postage for disseminating results.....	50
f. Incidental costs (reproduction, etc.).....	100
Total.....	\$6,400

CHAPTER IV

Analysis of Data

This study was conducted to explore the incidence of oxygen desaturation during transport from the operating room to the post anesthesia care unit in spinal anesthesia patients who received midazolam sedation. A convenience sample of 65 subjects was drawn from patients presenting for elective lower extremity orthopedic surgery at a community hospital in central Texas. Findings of the study are presented in this chapter.

Description of Sample

A total of 65 patients were initially entered into the study. Fourteen patients were dropped from data analysis. Two subjects did not meet inclusion criteria (64 year old and BMI of 37.8). General anesthesia was induced in one subject due to a block higher than the T-4 dermatome which resulted in profound refractory bradycardia. Ten subjects received additional sedative or analgesic agents and were eliminated from the study. One patient was eliminated due to equipment malfunction during transport. The final sample consisted of 51 patients. Demographic characteristics of the sample are summarized in

Table 2

Findings

Student's t -test was used to compare the means of the data sets. Pearson's correlation coefficient and linear regression were used to analyze the relationship between the variables.

Table 2

Patient Characteristics. Mean (SD) and Range. (n=51)

Age, years	30.84 (9.27) 18-50
Gender, M/F	44/7
Body mass index	26.06 (3.03) 18.4-34.1
ASA status	1 = 23
	2 = 28
Smoker	no = 30
	yes = 21
	pack-years: 3.03 (5.58) 0-30
Race	Black - 19
	Hispanic - 3
	Caucasian - 29
Midazolam dose (mg/kg)	0.05 (± 0.02) 0.01-0.1
Time from last dose of midazolam (min)	61.8 (± 32.4) 15-130
Dermatomal level	T7 (± 3.5) L5-T2
Percent SpO ₂ change	-0.88 (± 1.3) -5-1.0
Transport time (sec)	107.5 (± 34.8) 35-178

Our study found no incidence of hypoxemia ($\text{SpO}_2 < 90\%$) in lower extremity orthopedic ASA 1 and 2 patients during transport from the operating room to the post anesthesia care unit following spinal anesthesia with midazolam sedation. There was, however, a statistically significant decrease in the SpO_2 from $98.9 (\pm 1.54)$ to $98.0 (\pm 1.20)$ ($t = -4.94$, $p \leq 0.001$) during transport from the operating room to the post anesthesia care unit (refer to Figure 4). However, this is not clinically significant as 95.0 (the lowest SpO_2 recorded) is considered a normal SpO_2 in humans breathing room air.

The baseline SpO_2 mean was $98.2 (\pm 1.38)$ compared to the low SpO_2 mean of $98.0 (\pm 1.20)$. These values were not statistically significant as evidenced by $t = 0.76$ and $p = 0.449$. Likewise the SpO_2 I mean of $98.90 (\pm 1.20)$ compared to the SpO_2 III mean of $98.8 (\pm 1.23)$ was insignificant with $t = 0.71$ and $p = 0.481$.

The dependent variable, percent change in oxygen saturation, was calculated by subtracting the low SpO_2 from SpO_2 I. Pearson's correlation coefficient and linear regression analysis were used to test the effects of midazolam dose, dermatomal level, transport time, pack-year history, time from last dose of midazolam, and total operating room time, on the dependent variable. Only time from the last dose of midazolam was found to have a statistically significant effect ($p = 0.035$) on the percent change in oxygen

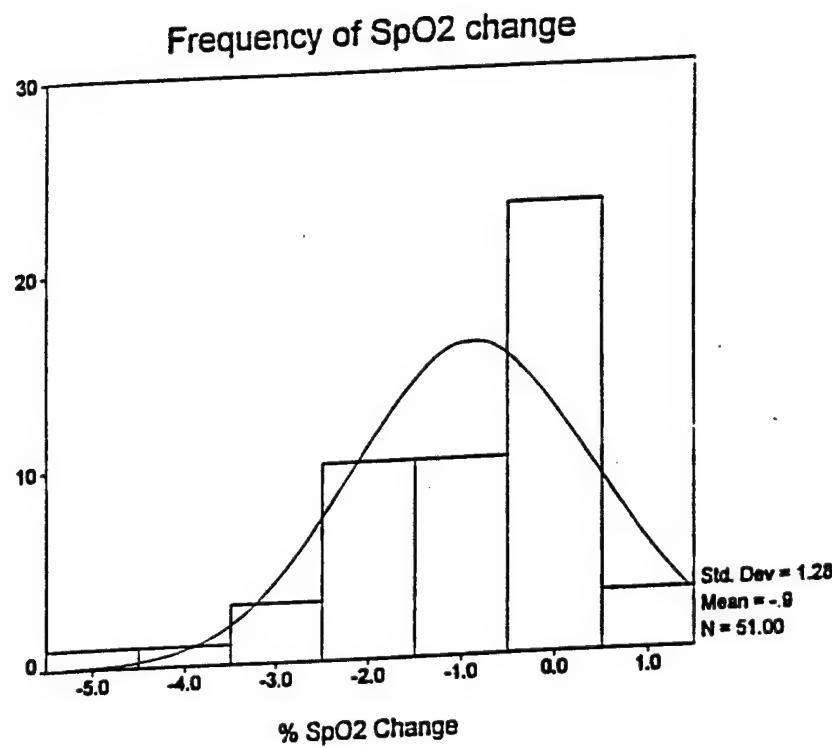


Figure 4. Frequency of SpO₂ change.

saturation. Based on the linear regression line the percent change in oxygen saturation is directly proportional to the time from the last dose of midazolam. The line is of poor fit to the data with $r^2 = 0.087$. While this anomaly in the data is interesting it is clinically irrelevant.

Study findings revealed that no clinically significant decrease in oxygen saturation occurred in lower extremity spinal anesthesia patients during transport from the operating room to the post anesthesia care unit. Improvement of SaO_2 during transport was noted in 6% of subjects, 43% maintained their SaO_2 saturations, and 51% experienced a decrease in their SaO_2 during transport from the operating room to the post anesthesia care unit.

CHAPTER V

Discussion, Conclusions, Implications, and Recommendations

Spinal anesthesia is generally considered a safe and effective alternative to general anesthesia. However, when spinal anesthesia is combined with midazolam sedation, patients may experience transitory respiratory depression or even death (Caplan et al., 1988). Currently there are no specific guidelines for transport of spinal anesthesia patients. In our institution, spinal anesthesia patients are transported by anesthesia care providers from the operating room to the post anesthesia care unit without oxygen or oximetry monitors. This practice may pose questions regarding patient safety during a critical postoperative period. While oxygen saturation during transport from the operating room to the post anesthesia care unit has been studied in general anesthesia patients (Kataria et al., 1988; Smith & Crul, 1988; Tyler et al., 1985), it has not been studied in spinal anesthesia patients. Our study determined that ASA 1 and 2 lower extremity orthopedic spinal anesthesia patients sedated with midazolam do not clinically desaturate during transport from the operating room to the post anesthesia care unit. In this chapter we discuss how the results support the conceptual framework, compare the findings with the literature review, identify strengths and limitations of the methodology, derive conclusions from supported data, identify implications for nursing, and recommend areas for further research.

Discussion

Our study examined the incidence of oxygen desaturation in adults during transport between the operating room and the post anesthesia care unit after spinal anesthesia with midazolam sedation. The conceptual frame work for our study was based on a physiological model of ventilation (see Figure1). Key factors identified in the conceptual framework were spinal anesthesia, sedation, and transport time between the operating room and the post anesthesia care unit. Each of these factors are addressed below.

The Student's *t*-test was used to compare the baseline SpO₂ (98.9) and the lowest SpO₂ (98.0). The statistic was *t* (51) = -4.94, *p* ≤ 0.001. While this small change in SpO₂ is statistically significant, it is not clinically significant, as 95.0 (the lowest SpO₂ recorded) is within the normal range of adult humans breathing room air. Our study defined clinically significant desaturation as < 90%. No patient demonstrated this level of significant oxygen desaturation during transport.

Spinal Anesthesia. Our study demonstrated no clinically significant desaturation during transport. The findings are in congruence with the findings of Yamakage et al. (1992) who found no change in SaO₂ in patients with a mean analgesic level of T6 ± 1.4 (*n* = 20). Gauthier et al. (1992) reported similar findings in nonsedated spinal anesthesia patients (T6 ± 1.0, *n* = 20). Both proposed an increase in rib cage contribution to ventilation and deafferentation of the chest wall as an explanation for a lack of desaturation. Loss of limb proprioceptor input has been theorized to result in desaturation during spinal anesthesia (Nadel et al., 1990). In our study the mean dermatomal level was T7 ± 3.5.

Dermatomal level of analgesia did not have a statistically significant effect on the decrease in SpO₂.

Midazolam Sedation. In our study the use of midazolam for sedation during spinal anesthesia did not demonstrate any clinically significant patient desaturation during the period monitored. The total dose of midazolam had no statistically significant effect on SpO₂. This differs from Halim et al. (1990) who theorized that the combination of spinal anesthesia and midazolam may seriously impair alveolar ventilation due to impaired function of intercostal respiratory muscles, decreased ventilatory response to hypoxia and inspiratory load. While we studied healthy ASA 1 and 2 patients, Halim et al. theorized that patients with significant pulmonary pathology would have a more profound impairment.

The study sample included 21 smokers. The average pack-year was 7.37 (± 6.6) pack-years. The lack of underlying disease processes, relatively young ages and minimal smoking histories of our population, possibly explains the lack of oxygen desaturation demonstrated in this study.

The time of last dose of midazolam was statistically significant for a negative change in SpO₂. As the time of last dose of midazolam increased, a negative change occurred in the SpO₂ measurement. Contrary to this, Curtis et al. (1989) demonstrated the peak decrease in SpO₂ (1.47%) occurred three minutes after midazolam administration in 31 patients without supplemental oxygen. This finding cannot be accounted for and is probably due to some mechanism which we did not measure.

Two factors in our study may explain the discrepancy between the mean low SpO₂.

Our patients received supplemental oxygen at two liters per minute via nasal cannula which would elevate the SpO₂ and increase the time before measurable desaturation occurs. The time range from the last dose of midazolam was 15 to 130 minutes, therefore the peak effect of the midazolam was well beyond the three minutes identified by Curtis et al. (1989).

Transportation. Currently there are no specific guidelines regarding transport of post spinal anesthesia patients to the post anesthesia care unit. To date a literature search reveals no published research to specifically address transport of spinal anesthesia patients.

It is difficult to compare general anesthesia and spinal anesthesia due to the ventilatory depressant effects of general anesthesia. For example, in a study of general anesthesia patients Smith and Crul (1988) reported a mean low SaO₂ of 92.8 % occurring at six minutes into transport time compared to our mean low SpO₂ of 98.02 \pm 1.54 for spinal anesthesia patients. Smith and Crul reported that the net change in saturation became significant at three minutes into transport time. However, our transport times were notably shorter with a mean of 107.5 \pm 34.8 seconds and a maximum of 178 seconds. Based on our findings we conclude that a clinically significant desaturation is not expected if transport time is less than three minutes.

Conclusions

1. No patients experienced desaturation ($\text{SpO}_2 < 90\%$) during transport from the operating room to the post anesthesia care unit.
2. A statistically significant decrease in oxygen saturation occurred with midazolam sedation and spinal anesthesia of ASA 1 and 2 patients undergoing lower extremity orthopedic surgery during transport from the operating room to the post anesthesia care unit. However, this decrease is not clinically significant.
3. Midazolam dose (0.01-0.1 mg/kg) and dermatomal level ($T7 \pm 3.5$) did not have a statistically significant effect on oxygen saturation.
4. It is safe to transport ASA 1 and 2 lower extremity orthopedic surgical patients with spinal anesthesia and midazolam sedation without supplemental oxygen to the post anesthesia care unit if transport time is less than three minutes.

Implications for Nursing

The results of this study verify the safety of transporting ASA 1 and 2 lower extremity orthopedic patients without oxygen from the operating room to the post anesthesia care unit. This may result in decreased costs by reducing usage of supplemental oxygen and supplies. Currently, there are no specific guidelines regarding supplemental oxygen and monitoring during transport to the recovery room for spinal anesthesia patients. This study may lend itself as a source of data or reference for determining supplemental oxygen and transport policies for similar type patients in perioperative settings.

Recommendations for Further Research

The scope of this study was limited to spinal anesthesia in ASA 1 and 2 patients with midazolam sedation during lower orthopedic procedures. The authors recommend further investigations with other sedative/hypnotic techniques, a sample of ASA 3 and 4 patients with significant pathology, interthecal narcotics, and perhaps different surgical procedures.

APPENDIX A

Informed Consent Form

VOLUNTEER AGREEMENT AFFIDAVIT

For use of this form, see AR 70-25; the proponent agency is OTSG

PRIVACY ACT OF 1974Authority:
10 USC 3013, 44 USC 1101, and 10 USC 1071-1087.

Principle Purpose: To document voluntary participation in the Clinical Investigation and Research Program. SSN and home address will be used for identification and locating purposes.

Routine Use: The SSN and home address will be used for identification and locating purposes. Information derived from the study will be used to document the study, implementation of medical programs, adjudication of claims and for the mandatory reporting of medical conditions as required by law. Information may be submitted to Federal, State and local agencies.

Disclosure: The furnishing of your SSN and home address is mandatory and necessary to provide identification and to contact you if future information indicates that your health may be adversely affected. Failure to provide the information may preclude your voluntary participation in the investigation study.

PART A(1) - VOLUNTEER AFFIDAVIT

Volunteer Subject in Approved Department of the Army Research Studies

Volunteers under the provisions of AR 40-38 and AR 70-25 are authorized all necessary medical care for injury or disease which is the proximate result of their participation in such studies.

I, _____, SSN _____, having full capacity to consent and having attained my _____ birthday, do hereby volunteer/give consent as legal representative for _____ to participate in _____

***Postoperative Oxygen Desaturation Following Spinal Anesthesia**

(Research Study)

under the direction of Vicki Nichols, BSN, MSN (MAJ, Army Nurse Corps) & Associate
Investigatorconducted at Darnall Army Community Hospital

(Name of Institution)

The implications of my voluntary participation; duration and purpose of the research study; the methods and means by which it is to be conducted; and the inconveniences and hazards that may reasonably be expected have been explained to me by:
Vicki Nichols BSN, MSN (MAJ, Army Nurse Corps)

I have been given an opportunity to ask questions concerning this investigational study. Any such questions were answered to my full and complete satisfaction. Should any further questions arise concerning my rights/the rights of the person I represent on study-related injury, I may contact:

Senior Judge Advocate at Darnall Army Community Hospital (317) 288-6307 or the Clinical Research Protocol Coordinator at Brooke Army Medical Center, Ft. SAM (210) 716-2598
(Name, Address and Phone Number of Hospital (Include Area Code))

I understand that I may at any time during the course of this study revoke my consent and withdraw/where the person I represent withdrawn from the study without further penalty or loss of benefit; however, the person I represent may be required (military volunteer) or requested (civilian volunteer) to undergo certain examinations if, in the opinion of the attending physician, such examinations are necessary for my/the person I represent's health and well-being. My/the person I represent's refusal to participate will involve no penalty or loss of benefits to which I am/the person I represent is otherwise entitled.

PART A(2) - ASSENT VOLUNTEER AFFIDAVIT (MINOR CHILD)

I, _____, SSN _____, having full capacity to consent and having attained my _____ birthday, do hereby volunteer for _____ to participate in _____

(Research Study)

under the direction of _____
conducted at _____ (Name of Institution)

(Continued on Reverse)

DA FORM 5303-R, MAY 88
IRS Approval Received 8 Aug 93

PREVIOUS EDITIONS ARE OBSOLETE

BEST AVAILABLE COPY

PART A(2) - ASSENT VOLUNTEER AFFIDAVIT (MINOR CHILD) (Cont'd)

The implications of my voluntary participation; the nature, duration and purpose of the research study; the methods and means by which it is to be conducted; and the inconvenience and hazards that may reasonably be expected have been explained to me by

I have been given an opportunity to ask questions concerning this investigational study. Any such questions were answered to my full and complete satisfaction. Should any further questions arise concerning my rights I may contact

at _____
(Name, Address and Phone Number of Hospital (Include Area Code))

I understand that I may at any time during the course of this study revoke my assent and withdraw from the study without further penalty or loss of benefits; however, I may be requested to undergo certain examination if, in the opinion of the attending physician, such examinations are necessary for my health and well-being. My refusal to participate will involve no penalty or loss of benefits to which I am otherwise entitled.

PART B - TO BE COMPLETED BY INVESTIGATOR

INSTRUCTIONS FOR ELEMENTS OF INFORMED CONSENT: (Provide a detailed explanation in accordance with Appendix E, AR 40-38 or AR 70-25).

TO: PARTICIPANTS IN THIS STUDY

*You are invited to participate in a research study of approximately 75 spinal anesthesia patients at Darnall Army Community Hospital. The study will involve determining the oxygen level in your blood after surgery when you are transferred from the operating room to the recovery room. This will help us to learn if patients should be transferred with oxygen to the recovery room after spinal anesthesia. If you choose to participate, your blood oxygen level will be read three times: 1) Before your operation begins 2) During transport from the operating room to the recovery room 3) After your operation in the recovery room.

Changes in blood oxygen levels have not been studied in spinal anesthesia patients during transport from the operating room to the recovery room. Since most patients don't routinely get oxygen during transfer, we believe it is an important area to investigate.

If you choose to participate, we will measure the oxygen level of your blood. To measure the oxygen level of your blood we will place a probe, very similar to the one you had during surgery, on your finger. This probe will be hooked up to a small machine/monitor that can measure the oxygen level of the blood. This probe will be on your finger from the time you leave the operating room to the time you enter the recovery room (approximately 2 to 5 minutes).

(CONTINUES ON ATTACHED PAGE)

I do do not (check one & initial) consent to the inclusion of this form in my outpatient medical treatment record.

SIGNATURE OF VOLUNTEER	DATE	SIGNATURE OF LEGAL GUARDIAN (If volunteer is a minor)
PERMANENT ADDRESS OF VOLUNTEER	TYPED NAME OF WITNESS	
IRB Approval Received 8 Aug 88	SIGNATURE of WITNESS	DATE

Potential Risks

This procedure is risk free and you will experience no discomfort as a result of your participation.

Potential Benefits

If we find your blood oxygen level to be low during transport from the operating room to the recovery room, you will be administered oxygen. We would not normally be aware of a drop during transport because blood oxygen level is not routinely monitored when a patient is transported from the operating room to the recovery room.

If you choose not to participate in this research study, it will in no way affect the medical/nursing care that you would normally receive. If you choose to withdraw at any time, this in no way will affect the medical/nursing care that you would normally receive. If you choose not to participate or withdraw from the research study at any time, no penalty or loss of benefits will occur.

You may be excluded from the study if your spinal anesthesia was unable to be maintained.

Point of Contact

If you have any questions, please ask us. If you think of any questions at a later time, please feel free to call the principal investigator of this study, MAJ Vicki Nichols, of the Department of Nursing, at (817) 288-8570. If you have questions about the ethical, legal or social aspects of this research, the review by the hospital Institutional Review Board (IRB) or other matters you would like to discuss with someone other than MAJ Nichols, you may contact the Clinical Investigation Protocol Coordinator at Brooke Army Medical Center (BAMC) (210) 916-2598 who will attempt to answer your questions or refer you to another appropriate person.

Confidentiality

Your name or any other personal identification will remain confidential. Only the research team will be able to identify you in relation to your study results. No research participants will be identifiable in any publication that may result from this study. All information will be presented as grouped data. However, your records may be reviewed by the BAMC Institutional Review Board and other regulatory agencies.

This research will take approximately one year to complete. Results of this study will be given to you upon request.

If you decide to participate, you will be given a copy of this consent form to take with you. Thank you for your time!

I have read the previous explanation and agree to participate in the research study described.

Patient's Initials

Date

Witness's Initials

Date

APPENDIX B**Data Collection Worksheet**

Oxygen Saturation After Spinal Anesthesia
Data Collection Form

Demographic data

Assigned study number: _____
 Age: _____ Gender: _____ Height: _____ Weight: _____ BMI: _____
 Smoker: _____ Pack/years: _____ Ethnicity: _____ ASA class: _____
 Surgical procedure: _____ Total OR time: _____
 SpO₂ alarm on the ProPaq 106 portable pulse oximeter set to alarm at 90%. Yes No
 Oxygen (500 psi minimum) and nasal canula on gurney prior to transport. Yes No

Preoperative measurements

Baseline SpO₂ prior to premedication or administration of the subarachnoid block _____
 Baseline vital signs: BP _____ HR _____ RR _____

Subarachnoid block data

Medication(s) and dose _____
 Patient position during insertion _____ Lumbar level of insertion _____

Postoperative measurements

Dermatome level of analgesia prior to removal of supplemental oxygen:

Left midaxillary line: _____ Right midaxillary line: _____

Temperature prior to transport _____

SpO₂I using the ProPaq 106 portable pulse oximeter _____

Total surgery time _____

Total dose of midazolam administered _____

Length of time from last dose of midazolam _____

Transport to post anesthesia care unit

Lowest SpO₂ during transport from the operating room to the post anesthesia care unit (SpO₂II) _____

Did the patient desaturate to an SpO₂ of 90% during transport? Yes No

If yes, note time 90% desaturation occurred _____

With the gurney in its final position in the PACU, prior to administering supplemental oxygen, what is the SpO₂ using the ProPaq 106 portable pulse oximeter? _____

Total transport time _____

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Vita

Vicki J. Nichols was born in Aitkin, Minnesota on May 19, 1961, the daughter of Reverend A. W. Carlson and Anne L. Carlson. She graduated from Aitkin High School, Aitkin Minnesota in 1979.

She received the degree of Bachelor of Science in Nursing from Saint Olaf College, Northfield, Minnesota in August 1983. In 1984, she received a direct commission into the U.S. Army Nurse Corps and is currently on active duty. She received the Master of Science degree in Nurse Management/Adult Health from the University of San Francisco in June 1992. In October 1995, she entered the U.S. Army/University of Texas Houston Health Science Center Program in Anesthesia Nursing. In 1987, she married Jeffrey C. Nichols.

Vita

David F. McCormick was born in Chicago, Illinois on September 1, 1957, the son of Edward D. McCormick Junior (deceased) and Anna Mae McCormick. After completing high school at Quigley Prep Seminary South, Chicago, Illinois in 1975, he enlisted in the Army and served three years on active duty. He continued his dedication to the military in the Army reserves until his return to active duty in 1990. He entered Richard J. Daley College and graduated in 1985 with an Associate Degree in Nursing. He completed his Bachelors Degree in Nursing in 1989 from Purdue University, Hammond, Indiana. In May of 1990, he was commissioned as a First Lieutenant. In October, 1995, he entered the U.S. Army/University of Texas Houston Health Science Center Program in Anesthesia Nursing. In 1990, he married Christine Courtney. They have two daughters, Deirdre born in 1991 and Brenna born in 1993.

Vita

Daryl J. Magoulick was born in Akron, Ohio on December 23, 1966, the son of Francis J. Magoulick and Ruth E. Magoulick. After completing his education at Arch Bishop Hoban High School in 1985, he attended the University of Akron. He received a Bachelors of Science in Nursing in 1990. Upon graduation, he was commissioned as a Second Lieutenant in the U.S. Army Nurse Corps and was stationed at Fort Knox, Kentucky. In October, 1995, he entered the U.S. Army/University of Texas Houston Health Science Center Program in Anesthesia Nursing. He married Lu Ann M. Henderson in June 1991.

Vita

Benjamin B. Simpson was born in Macon, Georgia on July 22, 1969, the son of Ben I. Simpson and Janice Simpson. He graduated from Southwest High School in 1987 in Macon, Georgia. He received a Bachelors of Science Degree in Nursing from Georgia College at Milledgeville, Georgia in 1992. He was married in 1992 to Angela Hamrick. In September 1992, he was commissioned in the U.S. Army Nurse Corps and entered active duty. He is currently enrolled in the U.S. Army/University of Texas Houston Health Science Center Program in Anesthesia Nursing with a graduation date of January 1998.

Vita

Phillip K. Haynie was born in Alexandria, Louisiana on June 6, 1970, the son of Larrie Haynie and Mary Thomas. After completing his work at Ringgold High School, Ringgold Louisiana, in 1988, he entered Northeast Louisiana University in Monroe, Louisiana. He received the degree of Bachelor of Science in Nursing in May, 1992. He was commissioned in the U.S. Army Nurse Corps and is currently on active duty. In October, 1995, he entered the U.S. Army/University of Texas Houston Health Science Center Program in Anesthesia Nursing. In 1995, he married Jane Wingerter.